Verticality perception in patients with lesions along the graviceptive pathways: acute deficits and subsequent compensation

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Abstract: Bilateral central vestibular pathways (CVP) send signals from the vestibular nuclei to the cerebellar, brainstem, and cortical areas that are involved in processing graviceptive signals. Whereas the estimated direction of gravity is accurate when upright, systematic angle-dependent errors can occur when roll-tilted: over-estimations at small and very large roll angles (E-effect) and roll under-estimation at medium-sized roll angles (A-effect). Acute lesions along the CVP frequently lead to deviations of the subjective visual vertical (SVV). Whereas SVV errors in upright positions have been well characterised, changes in the A- and E-effect and in SVV precision due to lesions along the CVP have not been studied in roll-tilted positions. We report on a series of patients with CVP lesions and compare SVV measurements in different roll orientations (0°, ±45°, ±90°) in the sub-acute state (4–33d) with follow-up 4 months later. In an upright position, 5/6 patients showed SVV deviations in the sub-acute state; in 3 patients deviations were ipsilesional. When rolltilted, 4/6 patients showed increased SVV errors. In all patients, the pattern of SVV errors could be explained by combining an SVV offset in an upright position with body-position-dependent errors when roll-tilted, being larger on the ipsilesional side and smaller on the contralesional side or vice versa. SVV precision was decreased in 4 patients. After 4 months, verticality perception was either improved (n = 1) or within normal range (n = 2) in terms of accuracy and precision in 3/4 patients. These results show that lesions along the CVP result in altered estimates of the direction of gravity in the entire roll plane, which can improve within a few months due to central compensation. At the time, accuracy had normalised in upright positions, estimated direction of gravity when roll-tilted could still be erroneous. Assessing the SVV in roll-tilted positions may reveal more subtle deficiencies and may hence support continuation of balance physiotherapy.

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Verticality perception in patients with lesions along the graviceptive pathways: acute deficits and subsequent compensation

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Abstract

Bilateral central vestibular pathways (CVP) send signals from the vestibular nuclei to cerebellar, brainstem, and cortical areas that are involved in processing graviceptive signals. Whereas estimated direction of gravity is accurate when upright, systematic angle-dependent errors occur when roll-tilted: over-estimations at small and very large roll angles (E-effect) and roll under-estimation at medium-sized roll angles (A-effect). Acute lesions along the CVP frequently lead to deviations of the subjective visual vertical (SVV). Whereas SVV errors in upright position have been well characterized, changes in the A- and E-effect and in SVV precision due to lesions along the CVP have not been studied in roll-tilted positions. We report on a series of patients with CVP lesions and compare SVV measurements in different roll orientations (0°, ±45°, ±90°) in the subacute state (4-33d) with follow-up ~4 months later. In upright position, 5/6 patients showed SVV deviations in the subacute state; in 3 deviations were ipsilesional. When roll-tilted 4/6 patients showed increased SVV errors. In all patients the pattern of SVV errors could be explained by combining an SVV offset in upright position with body-position dependent errors when roll-tilted, being larger on the ipsilesional side and smaller on the contralesional side or vice versa. SVV precision was decreased in 4 patients. After 4 months, verticality perception was either improved (n=1) or within normal range (n=2) in terms of accuracy and precision in 3/4 patients. These results show that lesions along the CVP result in altered estimates of direction of gravity in the entire roll plane that improve within few months due to central compensation. At the time accuracy had normalized in upright position, estimated direction of gravity when roll-tilted could still be erroneous. Assessing the SVV also in roll-tilted positions may reveal more subtle deficiencies and may hence support continuation of balance physiotherapy.

Introduction

The central vestibular pathways (CVP) interconnect a network of brainstem, cerebellar, thalamic, and cortical areas involved in the processing, integration, and perception of "graviceptive" input [1, 2]. Otolith and semicircular canal (SCC) inputs converge at the vestibular nuclei [3], project via bilateral ascending pathways through the medial lateral fascile (MLF) to the interstitial nucleus of Cajal (INC) and to the posterolateral thalamus, [4] and end in the temporo-peri-Sylvian vestibular cortex (TPSVC). The TPSVC is considered the human analogue of the parieto-insular vestibular cortex (PIVC) in non-human primates [5], where vestibular input converges with other sensory signals and subserves perception of spatial orientation and navigation [6]. In the upright position, the internal representation of gravity and spontaneous head orientation aligns with gravity, while the torsional orientation of the eyes is symmetric (binocular fundoscopic torsion = 0°). Depending on the exact topography of the lesion, vestibular tone-imbalances lead to roll tilts of perception, head, and body as well as to misalignments of the visual axes (skew deviation) [7, 8] and binocular torsion [1].

Perception of self orientation relative to gravity can be assessed by aligning an illuminated line with the perceived vertical, termed the "subjective visual vertical" (SVV) [9]. Under static conditions in darkness the SVV is mainly influenced by otolith signals. Estimates of SVV in upright are accurate (i.e., within a range of ± 2°, [9, 10]). Aligning a luminous line to perceived vertical while roll-tilted requires the subject to compensate for body roll. Body roll leads to errors towards over-estimation (E-effect) at moderate roll angles up to 60° [11-13], whereas at roll angles larger than 60° perceived body roll is biased towards under-estimation (A-effect) [12, 14]. It was suggested that A- and E-effects are a consequence of how various sensory signals are integrated into a unified percept of vertical [14]. While Mittelstaedt put a focus on an imbalanced otolith input to cause the A- and E-effects [14], more recent modeling of perceived vertical underlined the role of a noisy but accurate otolith signal [13, 15]. To maximize the precision of verticality estimates, this noisy otolith estimate is combined with a bias that represents prior knowledge about where vertical is located. This bias refers to the body-longitudinal axis and is based on the assumption that small roll-tilt angles are more likely than large angles. Using this modeling approach, verticality estimates at small roll angles are accurate and precise, however, at larger roll angles, both A- and E-effects arise, reflecting the experimental data well [13, 15].

Following unilateral or bilateral lesions along the CVP, the SVV becomes biased and increased noise levels of the centrally obtained estimate of vertical will interfere with the A-

and E-effect. However, little is known about the interaction between the A- and E-effect and shifts caused by lesions along the CVP. Hypothetically, various changes in the pattern of A- and E-effects may emerge. Most straight forward is the hypothesis that an imbalance of the graviceptive input is combined additively with the physiologic deviations when roll-tilted, resulting in a *constant offset* of the A- and E-effect over the entire roll plane (hypothesis 1). Alternatively, shifts in SVV in upright position may be accompanied by body-position dependent errors in the direction or opposite to the direction of the SVV offset in upright position. Thereby E-effects would become more pronounced and A-effect would decrease (hypothesis 2) or vice versa (not shown). Overall, shifts in the SVV may result in larger A- and E-effects as the noise level of the graviceptive signal is likely larger. Under these circumstances, the brain might put more weight on prior knowledge at the cost of *increased SVV errors* at larger roll angles (hypothesis 3).

/* Figure 1 about here*/

In patients with acute lesions along the CVP, verticality perception has been studied in upright position only. Whereas peripheral vestibular [16-19] and caudal brainstem lesions resulted in ipsiversive tilts of SVV, more rostral brainstem lesions yield contraversive SVV deviations [20]. Contraversive SVV tilts and contraversive signs of ocular tilt reaction (OTR; consisting of ocular torsion, skew deviation and head-tilt) in patients with acute cerebellar stroke have been related to either nodular [21] or dentate nucleus lesions [22], whereas in patients with ipsiversive SVV tilts and ipsiversive OTR the dentate nucleus was spared and lesions were located in the biventer lobule, the middle cerebellar peduncle, the tonsil and the inferior semilunar lobule [22]. Acute lesions within the territory of the middle cerebral artery including the posterior part of the insula, the insular gyrus, and the middle and superior temporal gyrus yielded mostly contraversive SVV tilts and imbalance of gait [23]. Ipsiversive SVV tilts and impaired postural stability was reported in a patient with a metastasis extending from the superior temporal gyrus to the first long insular gyrus [24], suggesting involvement of the superior temporal gyrus in estimating gravity.

Here we evaluated both the accuracy and precision of SVV adjustments in patients with lesions along the CVP in various roll-tilted positions both in the subacute and the chronic state to further characterize 1) the pattern of A- and E-effects when the graviceptive input is imbalanced and 2) recovery of verticality perception both in terms of accuracy and precision.

Based on the literature, we hypothesized that both the pattern of roll over- and under-estimation and precision are altered in the patients.

Methods

Subjects

We report on a series of patients with acute ischemic or hemorrhagic lesions along the CVP treated at the Department of Neurology at the University Hospital Zurich between November 2007 and December 2008 and compare measurements in the subacute stage with follow-up ~4 months later (obtained in 4 out of 6 patients). Inclusion criteria were clinical findings suggestive of an acute lesion along the CVP (e.g. spontaneous or gaze-evoked nystagmus, partial or complete ocular tilt reaction, gait ataxia, dysmetria) or neuro-imaging findings suggestive of an affection of the CVP. The locations of the lesions are described in the single cases. Informed written consent of all subjects was obtained after a full explanation of the experimental procedure. The protocol was approved by a local ethics committee and was in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki for research involving human subjects.

Experimental setup

In all patients a careful neuro-ophthalmological and neuro-otological examination was performed. Magnetic resonance imaging (MRI) including Flair, T2- and diffusion-weighted imaging (DWI) was obtained in all patients. SVV adjustments were obtained on a motor-driven turntable (Acutronic, Jona, Switzerland). Subjects were secured with a 4-point safety belt and the head was fixated viewing straight ahead with a thermoplastic mask. Five whole-body roll orientations were studied: upright, 45° right-ear down (RED), 90° RED, 45° left-ear down (LED), and 90° LED. A remote control box allowed the subjects to rotate an arrow projected on a sphere in front and to confirm adjustments. The arrow projection started 10 seconds after the turntable came to a full stop. Arrow starting position deviated pseudo-randomly between 28 and 72° clockwise (CW) or counter-clockwise (CCW). Subjects were instructed to adjust the arrow along the perceived gravitational vertical (gravicentrically) in otherwise complete darkness within 15 seconds. After a training session each trial type was run 24 times, resulting in a total of 120 trials, recorded in a single session. For static SVV adjustments as used here we have previously checked for post-rotary torsional ocular drift and nystagmus to quantify the contribution of SCC stimulation after the movement and showed

that average torsional eye velocity at the time subjects confirmed arrow adjustments was small (0.10 ± 0.06°/s) [25]. For comparison, we considered SVV adjustments previously obtained from a group of seven healthy normal subjects using a similar paradigm [15].

Data analysis

SVV adjustment outliers were defined as data points differing more than three standard deviations (SD) from the mean. In total, less than 0.1% of all trials were discarded. Average deviations relative to earth-vertical and the SD were calculated for each subject. CW deviations relative to earth-vertical have a positive sign. In the following, we will use the term "trial-to-trial variability" whenever we report intra-individual SD. One of the co-authors, an experienced neuroradiologist (SB) delineated the location and extension of the lesions and the exact involvement of the CVP on all MRI sequences obtained.

Results

Description of single cases

Patient 1 (DD): This 33 year old male patient presented with slurred speech, acute right hemiataxia, and gait deviation to the right. No spontaneous or direction-changing horizontal nystagmus was noted, pursuit was smooth and saccades were metric. On MR-imaging right cerebellar ischemia in the territory of the superior cerebellar artery (SCA) affecting the right superior cerebellar peduncle, the biventer lobe and the inferior semilunar lobe, but sparing the nodulus, the dentate nucleus and midbrain structures was found. In the subacute stage (recorded 10 days after initial symptoms) the SVV was tilted ipsilesionally (i.e., CW) by 5.4° on average when upright. In all roll-tilted positions, roll over-estimation (E-effect) was noted; however, deviations were clearly larger for LED positions compared to RED positions. In the follow-up recovery was noted, SVV estimates in upright position were accurate. In roll-tilted positions SVV deviations were symmetric (RED vs. LED) and consistent with the literature (variable E-effects at small roll angles, A-effects at roll angles > 60° ear-down). Overall, this patient showed a tendency towards roll over-estimation in all roll-tilted positions. Trial-to-trial variability in both the subacute and the chronic stage was not affected and resembled the pattern observed in the control subjects.

Patient 2 (EW): This 66 year old male patient presented with acute dysarthria, right-sided dysmetria, direction-changing horizontal nystagmus, and gait ataxia. MR-imaging showed a

right cerebellar hemorrhage including the brachium pontis, the dentate nucleus and the superior cerebellar peduncle on the right side. SVV adjustments (obtained 5 days after onset of symptoms) showed an average contralesional (i.e., CCW) tilt of SVV (-10.7°) in upright position. In all roll-tilted positions large roll over-compensation with deviations up to 60° were noted. In the follow-up SVV in upright position was accurate and slightly reduced, but still considerably large E-effects persisted after 4 months in all roll-tilted positions. Trial-to-trial variability was increased in all roll positions studied in the subacute stage. The roll-angle dependent modulation of SVV variability (yielding increasing values with increasing roll angle), however, was reduced. Compared to the subacute stage, variability was slightly reduced in the follow-up, however, remaining clearly larger than the healthy controls.

Patient 3 (MK): This 65 year old male patient presented with vertical gaze palsy, slow upward saccades, abolished downward saccades, missing torsional quick-phases when tilting the head to the right, hypophonia, hypersomnia, and right hemiataxia. MR-imaging yielded a paramedian central thalamic lesion on the left side and a paramedian rostral thalamic lesion on the right side. On the left side the lesion expanded into the midbrain affecting the medial longitudinal fasciculus (MLF) and the interstitial nucleus of Cajal (INC). SVV adjustments (obtained 19 days after onset of symptoms) yielded an average CCW tilt (-6.5°) in upright position, which is ipsilateral to the left paramedian mesencephalic lesion. In roll tilted positions, under-compensation was either reduced (in 90° RED) or inversed, now yielding roll over-compensation (90° LED). In the follow-up, the pattern of SVV changed: now an average offset of 3.6° into CW direction while being upright was found and in roll-tilted positions, SVV adjustments were shifted CW relative to the healthy control subjects. This shift was larger for RED roll-tilts. Trial-to-trial variability was several fold increased in the subacute stage (being most prominent in 45° LED position) and partially normalized after ~4 months, however, remaining at a clearly larger level than in the control subjects.

Patient 4 (FK): This 65 year old male patient presented with horizontal gaze palsy, impaired upgaze, left trigeminal hypesthesia, and left-sided sensorimotor impairment. On initial MR-imaging a predominantly right-sided ponto-mesencephalic dorsal hemorrhage was found, including the medial vestibular nuclei bilaterally, the superior and the lateral vestibular nucleus on the right side, the INC on the right side and the MLF on both sides. No cerebellar (e.g. affecting the dentate nucleus or the cerebellar nodulus) or thalamic structures were involved. At follow-up after four months, the patient newly developed a vertical pendular

nystagmus and increasing gait ataxia. Repeated MR-imaging revealed a hypertrophic degeneration of the right inferior olive. SVV adjustments obtained 16 days after onset of symptoms in upright position deviated ipsilesionally (3.7° on average) whereas in roll-tilted positions roll under-compensation – being clearly increased compared to the healthy controls – was noted. At follow-up large contralesional SVV deviations in upright position (averaging -15.2°) were found. Trial-to-trial variability at follow-up remained increased and changed little compared to the initial evaluation.

Patient 5 (UM): This 59 year old male patient presented with vertical diplopia worsening when looking to the right, hypersomnia, and attention deficits. On MR-imaging a subacute ischemic lesion within the right paramedian anterior thalamus and within the left paramedian posterior thalamus was found. No mesencephalic involvement could be depicted. We hypothesized that the diplopia was due to a central left-sided fourth nerve palsy caused by a transient compression of the left midbrain resulting from edema of the left paramedian thalamic infarction. The first assessment of subjective vertical in this patient was delayed (33 days after onset of symptoms) and within the normal range, showing no offsets of SVV in upright position. In this patient no follow-up SVV measurements were obtained.

Patient 6 (SJ): This 61 year old male patient initially presented with hypersomnia and impaired cognition. On MR-imaging a left-sided thalamic lesion affecting both the central and rostral paramedian thalamus and the posterolateral thalamus (including the nucleus ventro-oralis intermedius [Vim] and the nucleus ventrocaudalis externus (Vce)] was found. There was no evidence for midbrain involvement. SVV measurements (obtained 4 days after the first clinical symptoms) showed contralesional, CW deviations (4.3° on average) in upright position, whereas at 90° ear down position (both RED and LED) deviations consistent with roll under-compensation were increased compared to the healthy control subjects. Trial-to-trial variability in upright position was within normal range, however, in roll-tilted positions precision of adjustments was clearly impaired. In this subject no follow-up SVV recordings could be obtained.

/*Figure 2 about here*/

Summary of findings

Five out of six patients showed shifts of SVV in upright position in the subacute stage, either into clockwise (4.5 ± 0.9°; mean ± 1 SD; trials with CW and CCW arrow rotations pooled) or counter-clockwise (-8.4 ± 3.3°) direction. In three out of these five patients, deviations were towards the side of the lesion whereas in the remaining two patients the shifts were contralesional. Whereas in the two patients with subacute cerebellar lesions (DD and EW) and in the patient with a left mesencephalic lesion (MK) including the MLF and the INC shifts from roll under-compensation towards roll over-compensation in roll-tilted positions were found, two patients (FK and SJ) yielded increases in roll under-compensation. In all these patients the pattern could be explained by combining an offset of SVV in upright position with body-position dependent changes in SVV errors in roll-titled positions shifting from A- towards E-effects or vice versa (yielding increasing A-effects). One patient with a bilateral paramedian thalamic lesion (UM) showed bilaterally intact CVP both clinically and when testing the SVV. However, in this patient SVV recordings were delayed by more than a month relative to the onset of complaints. An impairment of the CVP in the earlier acute / subacute stage therefore cannot be excluded in this patient, however, based on the MR-imaging (showing no signs of posterolateral thalamic or mesencephalic involvement) would be unexpected. Precision of adjustments was decreased in four out of six patients (EW, MK, FK, and SJ), but the pattern of modulation (i.e., increased trial-to-trial variability with increasing whole-body roll tilt) was preserved.

In the follow-up SVV measurements approximately four months later, verticality perception in upright and roll-tilted positions was either improved (n=1) or had normalized completely (n=2) in terms of accuracy and precision in three out of four patients. One patient with a ponto-mesencephalic dorsal hemorrhage (FK), however, showed worsening of both clinical symptoms and SVV errors at follow-up. Precision of adjustments remained poor. We attributed these findings to a newly developed hypertrophic degeneration of the right inferior olive.

Discussion

The found changes of SVV deviations in the subacute stage of patients with lesions along the CVP suggest that body-position dependent changes of SVV errors when roll-tilted occur on top of an SVV error in upright position. This result is most consistent with hypothesis 2 (see Fig. 1). Whereas in patients with cerebellar and mesencephalic lesions deviations were shifted towards roll over-compensation (E-effect), increasing roll under-compensation (A-effect) was noted in one patient with a ponto-mesencephalic (FK) and in

one patient with a unilateral posterolateral and paramedian thalamic (SJ) lesion. Such increases or decreases of A-effects and shifts towards E-effects over the entire roll plane are likely related to the lesioned CVP and suggest altered central processing of graviceptive input. Considering the often inhibitory function of vestibulo-cerebellar structures the observed E-effects in patients with cerebellar lesions could be interpreted as loss of cerebellar inhibition. In the patient with a paramedian mesencephalic lesion (MK) the increased E-effects could be related to impaired processing of inhibitory cerebellar input. In the two patients with increased A-effects a stronger impact of inhibitory cerebellar input could hypothetically provide an explanation.

We observed both ipsi- and contralesional deviations of the SVV in upright position. Similarly, the direction of the deviations (ipsilesional vs. contralesional) varied also in the two patients with unilateral cerebellar lesions and likely depends upon the location of the cerebellar lesion. Whereas a contralesional shift was noted when the dentate nucleus was affected (EW), an ipsilesional shift was observed when this structure was spared (DD). Previously contraversive SVV tilts in patients with acute cerebellar stroke have been related to lesions of dentate nucleus [22] or the cerebellar nodulus [21], whereas in patients with ipsiversive SVV tilts lesions of the biventer lobe, the inferior semilunar lobe, the tonsils and the middle cerebellar peduncle were noted, with the dentate nucleus being spared [22]. These descriptions match our observations well.

Within the brainstem, it was proposed that more caudal lesions (ponto-medullary) yield ipsiversive SVV deviations whereas more cranial lesions (ponto-mesencephalic) result in contraversive SVV tilts [1, 20]. Here we noted ipsilesional SVV shifts in a patient with a mesencephalic lesion including the MLF and the INC in the subacute stage (MK), which is opposite previous observations. Noteworthy, in this subject SVV deviations shifted over time, being contralesional at follow-up. Possibly the direction of SVV deviations in mesencephalic lesions vary over time and may be ipsilesional in earlier stages. In another patient (FK) with a ponto-mesencephalic hemorrhage, we observed an ipsilesional shift in the subacute stage, which would be in agreement with the findings from Brandt and Dieterich.

Whereas posterolateral thalamic lesions presented with deviations of SVV in a majority of cases in a study reported by Dieterich and Brandt, deviations in perceived direction of gravity in patients with anterior paramedian thalamic lesions were linked to additional midbrain tegmental involvement [4]. In the two patients with paramedian thalamic lesions studied here, one (SJ) showed a contralesional shift, which was associated with posterolateral thalamic involvement (including the Vim and Vce) on MR-imaging. Both the

Vim and Vce have been reported to process graviceptive signals, yielding ipsi- or contraversive SVV tilts without concomitant OTR [4]. The other patient (UM) initially had signs of a midbrain involvement (vertical diplopia on lateral gaze, possibly associated with a nuclear trochlear nerve palsy), however, when recording the SVV in this patient (with a delay of 33 days after symptom onset) we noted intact perception of verticality along with diminished vertical diplopia. In this subject we hypothesize that the thalamic lesion transiently compressed midbrain structures, leading to a nuclear fourth nerve palsy and explaining the vertical diplopia. On MR-imaging this patient presented with no involvement of midbrain structures that belong to the CVP (as the MLF and the INC).

The decreased precision of adjustments noted in four patients reflects more uncertainty about self-orientation in space due to impaired central processing of bilateral vestibular input. Improvements of accuracy and precision of verticality perception within four months after lesions along the CVP demonstrate that the body-position dependent SVV errors observed in the subacute stage are readjusted, most likely as a result of central compensation. However, as shown in a patient with a dorsal ponto-mesencephalic hemorrhage and hypertrophic degeneration of the right inferior olive (Mollaret degeneration) at follow-up (see [26] for review), interruption of the rubro-dentato-olivary tract (termed 'Guillain-Mollaret triangle' [27]) can interfere with recovery.

Evaluation of internal estimates of direction of gravity both in terms of accuracy and precision is a sensitive means to quantify the integrity of the CVP. During rehabilitation patients with lesions along the CVP may first regain their ability to estimate direction of gravity when upright. However, they may still demonstrate difficulties (as reflected in increased A- or E-effects and larger trial-to-trial variability) in the same task when roll-tilted. When assessing the patient's behavioural skills with regards to perceived direction of gravity, restriction to upright position may under-estimate residual symptoms and may therefore lead to premature cessation of rehabilitative efforts. Based on our data, we propose assessing the integrity of the CVP in various roll-tilted positions also.

Figure legends

Figure 1:

In this figure distinct hypothetical changes in perceived vertical in both upright and roll-tilted positions are illustrated (grey circles). For comparison average SVV adjustments from healthy normal subjects (in black) are shown. Whereas hypothesis 1 suggests a constant offset of SVV over the entire roll plane, hypothesis 2 proposes body-position dependent SVV errors in roll-tilted positions that are into the same direction (shown) or into the opposite direction (not shown) as the offset of SVV in upright position. Alternatively, lesions along the CVP may increase the noise in the sensory estimate and cause not only a shift of SVV in upright but increase the error signal, i.e., both established A- and E-effects would become larger (hypothesis 3).

Figure 2:

Single subject average SVV errors (left column) and trial-to-trial SVV variability (right column) are plotted against whole-body roll angle, both in the subacute stage (in dark-grey) and for the follow-up ~4 months later (in light grey). Trials with CW (circles) and CCW (squares) arrow rotations are shown separately. For comparison, average (±1 SD) SVV errors and variability values from healthy control subjects [15] are shown (in black). Note that the scaling on the ordinate for the SVV accuracy (subject 2) and SVV precision (subjects 2 and 3) is different.

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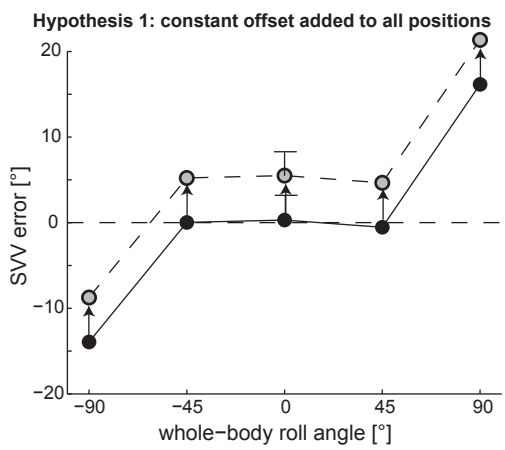
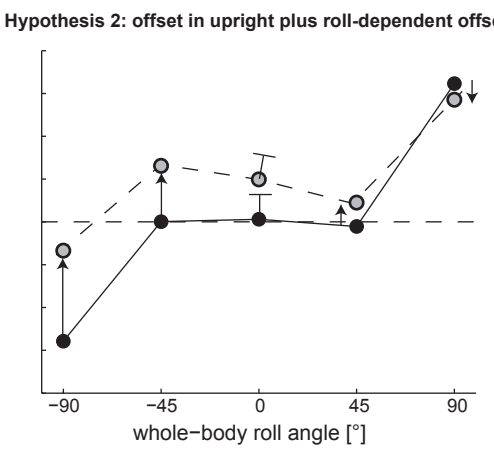
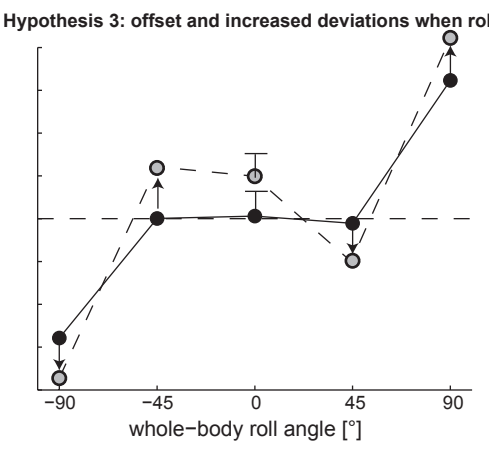
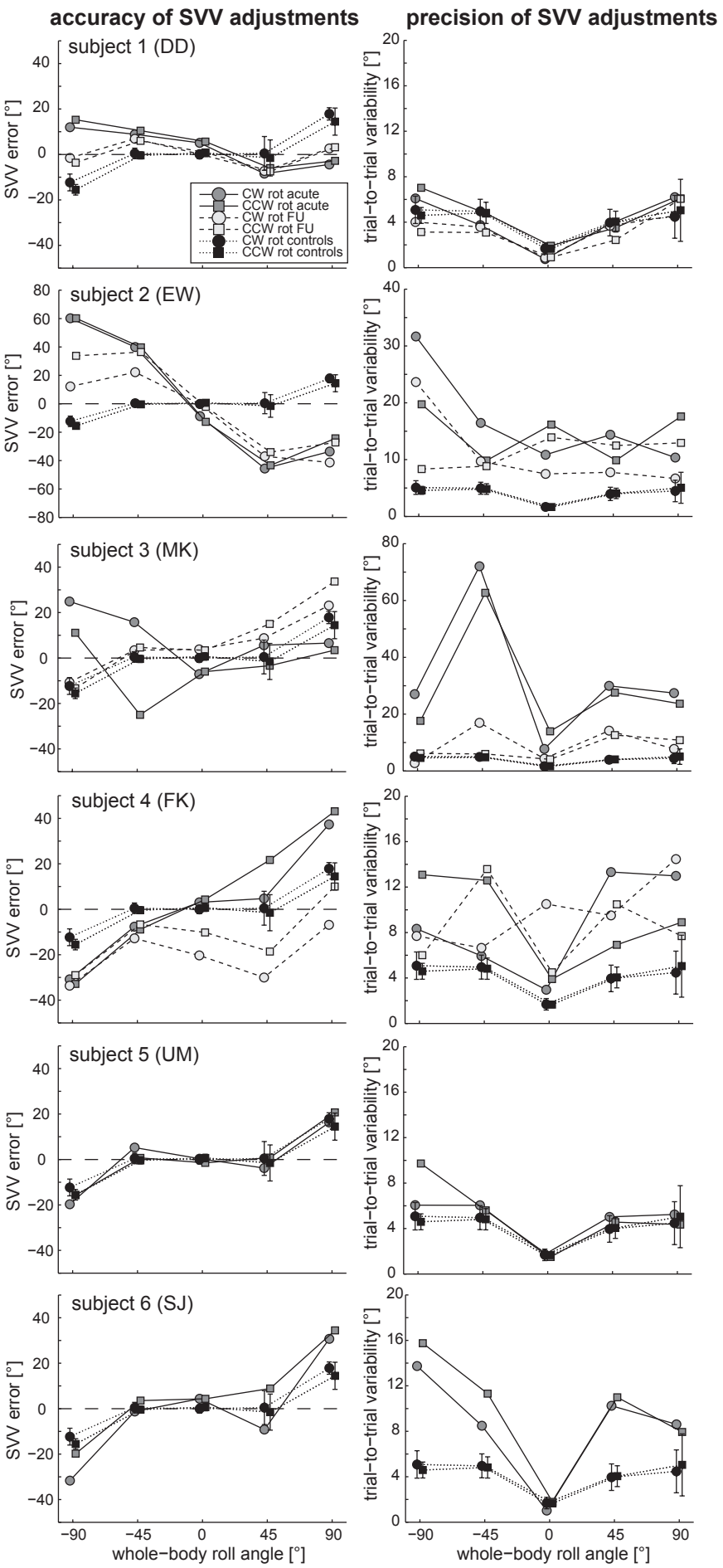

Hypothesis 1: constant offset added to all positions
SVV error [°]
20
10
0
−10
−20
−90
−45
0
45
90
whole−body roll angle [°]


Hypothesis 2: offset in upright plus roll-dependent offset
−90
−45
0
45
90
whole−body roll angle [°]


Hypothesis 3: offset and increased deviations when rolled
−90
−45
0
45
90
whole−body roll angle [°]


accuracy of SVV adjustments
precision of SVV adjustments
subject 1 (DD)
subject 2 (EW)
subject 3 (MK)
subject 4 (FK)
subject 5 (UM)
subject 6 (SJ)
SVV error [°]
trial−to−trial variability [°]
CW rot acute
CCW rot acute
CW rot FU
CCW rot FU
CW rot controls
CCW rot controls
−90
−45
0
45
90
whole−body roll angle [°]